

pathway. This work has important implications for the design of new strategies to control NLRP3-related disease.

## GW26-e0768

### Ticagrelor Prevents Cardiac Inflammation and Fibrosis of Hypertension Rats

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**OBJECTIVES** In this study, we aimed to determine the P2Y<sub>12</sub> receptor inhibitor ticagrelor could inhibit cardiac inflammation and fibrosis of hypertension rats.

**METHODS** Male Sprague Dawley rats were made hypertension after partial renal artery constriction (2-kidney, 2-clip method). After 1 week, they were simultaneously treated with ticagrelor (10 mg/kg ig.q24h) or vehicle. At 8 weeks, echocardiographic measurements were taken to observe structure and blood flow in heart of rats; CD41 staining showed that platelets accumulation at heart of rats; Real-time PCR was used to measure inflammatory cell infiltration into the heart being responsible for cardiac fibrosis.

**RESULTS** At 1, 4, 8 weeks systolic blood pressure (mmHg) were increased in hypertension rats. Ticagrelor or vehicle did not change the systolic blood pressure (mmHg) of hypertension rats. CD41 staining showed that platelets accumulated at heart of vehicle-treatment hypertension rats was more than normal rats. Ticagrelor treatment hypertension rats had significantly decreased accumulation of  $\alpha$ -SMA(+) myofibroblasts and cardiac fibrosis, compared to vehicle hypertension rats. Inflammatory cells: Mac-2(+) macrophages and CD45(+)Ly6G(+) neutrophils in heart were also inhibited at ticagrelor treatment hypertension rat, compared to vehicle hypertension rats. Interleukin-1 $\beta$  and transforming growth factor- $\beta$  examined by Real-time PCR and immunohistochemical staining was significantly decreased in heart of ticagrelor treatment hypertension rats, compared vehicle hypertension rats in heart.

**CONCLUSIONS** Our data suggested that ticagrelor ameliorates cardiac inflammation and fibrosis of 2-kidney, 2-clip method hypertension rats.

## GW26-e1325

### Dynamic changes of Serum myocardial enzymes in 73 infantile cases with rotavirus gastroenteritis

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**OBJECTIVES** To investigate Dynamic changes of serum myocardial enzymes in children less than three years of age with rotavirus gastroenteritis.

**METHODS** A total of 73 patients were enrolled in observation group. All had acute gastroenteritis due to rotavirus infection and were treated at Affiliated Hospital of Sun Yat-Sen University between January 2013 and March 2015. All were younger than 3 years of age (39 males and 34 females aged 0.2 to 3 years, median 17 months), and those patients with congenital heart disease, myocarditis, liver and kidney diseases, muscular diseases, toxic encephalopathy and epilepsy must be excluded. All of the patients had watery diarrhea, stool rotavirus antigen were positive. Patients were allocated to two groups according to with or without Symptoms of dehydration (38 patients in dehydration group and 35 patients in non- dehydration group). Blood samples of all patients were collected on day 1 and day 7 for detection of Serum myocardial enzymes, including creatine phosphate kinase (CK), creatine phosphate kinase isoenzyme (CK - MB), aspartic transaminase (AST), and lactate dehydrogenase(LDH). 36 healthy children aged 0.5 to 3 years in the same period were taken as control whose blood samples were collected on day 1. In addition to basic treatment, those patients with abnormal Myocardial enzyme were treated with fructose 1,6-diphosphate and high-dose vitamins C.

**RESULTS** Serum enzymes levels (CK, CK - MB, AST and LDH) on day1 in observation group were higher than those of control group, and the difference was statistically significant ( $P < 0.05$ ). Both CK and CK-MB increased among them accounted for 38.3% of cases. The percentage of abnormal enzymes (both CK and CK-MB) in dehydration group was significantly higher than that of non- dehydration group (46% VS 31%), and the difference was significant ( $P < 0.05$ ). With the condition of the patients improved, Serum myocardial enzymes levels on day 7 after

treatment was significantly decreased. The difference was statistically significant ( $P < 0.05$ ) compared with the levels on day 1, and was not statistically significant ( $P > 0.05$ ) compared with the control group.

**CONCLUSIONS** The patients younger than 3 years with rotavirus gastroenteritis were susceptible to develop myocardial damage. Consequently early detection and treatment are recommended.

## GW26-e2222

### Polymorphisms in the DOCK7 gene and the risks of coronary artery disease and ischemic stroke

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**OBJECTIVES** Several recent genome-wide association studies in different populations have identified the *DOCK7* genetic variants influencing serum lipid levels, but the results are inconsistent. In addition, it is still unclear whether these loci identified also exert the similar effect on the susceptibility of coronary artery disease (CAD) and ischemic stroke (IS). Therefore, the present study aimed to detect the association of the *DOCK7* single nucleotide polymorphisms (SNPs) and serum lipid levels, the susceptibility of CAD and IS in the Guangxi Han population.

**METHODS** This study recruited 1,139 unrelated patients (CAD, 584 and IS, 555) and 627 healthy controls from the First Affiliated Hospital, Guangxi Medical University. The diagnosis of CAD was based on typical clinical symptoms, electrocardiographic changes, increased serum markers including creatinine kinase-MB and troponin T, and coronary angiographic findings (coronary stenosis  $\geq 50\%$  in at least either one of the three main coronary arteries or their major branches such as diameter  $\geq 2$  mm). The classification of IS was made according to the TOAST (Trial of Org 10172 in Acute Stroke Treatment) criteria. Genotypes of the *DOCK7* rs10889353 and rs10889335 SNPs were determined by the Snapshot technology platform.

**RESULTS** Serum total cholesterol (TC) and triglyceride (TG) levels in healthy controls were different among the three genotypes of *DOCK7* rs10889353 and rs10889335 SNPs ( $P < 0.05$ -0.01), the rs10889353C and rs10889335G allele carriers had higher TC and TG than the C and G allele non-carriers; respectively. The rs10889353C and rs10889335G allele carriers were associated with an increased risk of CAD (rs10889353AC genotype: OR = 1.20, 95%CI = 0.94-1.54,  $P = 0.079$ ; CC genotype: OR = 2.76, 95%CI = 1.49-5.14,  $P = 0.001$ ; rs10889335AG genotype: OR = 1.23, 95%CI = 0.96-1.57,  $P = 0.061$ ; and GG genotype: OR = 2.44, 95%CI = 1.35-4.43,  $P = 0.002$ ). The rs10889353C allele carriers were also associated with an increased risk of IS (AC genotype: OR = 1.12, 95%CI = 0.87-1.44,  $P = 0.202$ ; CC genotype: OR = 2.25, 95%CI = 1.18-4.28,  $P = 0.009$ ). After adjustment for age, gender, body mass index (BMI), smoking, drinking, hypertension, hyperlipidemia and diabetes, the rs10889353 SNP was still associated with an increased risk of CAD and IS in different genetic models ( $P < 0.05$ -0.01). Stratified analysis showed that the two SNPs may interact with the gender, age, BMI, smoking, drinking, hypertension and hyperlipidemia to affect (increase or decrease) the risks of CAD and IS.

**CONCLUSIONS** *DOCK7* rs10889353 and rs10889335 SNPs are associated with elevated serum TC and TG levels, and increased risk of CAD in the Guangxi Han population. The rs10889353C and rs10889335 G allele carriers have higher serum TC and TG levels and higher risk of CAD than the rs10889353AA and rs10889335AA homozygotes. *DOCK7* rs10889353 SNP is also associated with the susceptibility of IS, the rs10889353CC homozygote is associated with an increased risk of IS.

## GW26-e2337

### Clinical significance of serum C-reactive protein and blood homocysteine detection in patients with coronary heart disease

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**OBJECTIVES** Coronary heart disease (CHD) is one of the most common heart disease, it is because of coronary artery stenosis, insufficient blood supply myocardial dysfunction and (or) caused by organic disease. To discuss the relationship between the serum C-reactive protein(CRP), blood homocysteine (Hcy) levels of coronary heart disease patients and coronary heart disease.